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SEA COX-2

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L3 ANSWER 1 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:103504 BIOSIS
DOCUMENT NUMBER: PREV200300103504
TITLE: Galphaq signaling is required for Rho-dependent transcriptional activation of the cyclooxygenase-2 promoter in fibroblasts.
AUTHOR(S): Slice, Lee W. (1); Han, Sang-kyou; Simon, Melvin I.
CORPORATE SOURCE: (1) University of California, 900 Veteran Avenue, Los Angeles, CA, 90095-1786, USA: lslice@mednet.ucla.edu USA
SOURCE: Journal of Cellular Physiology, (February 2003, 2003) Vol. 194, No. 2, pp. 127-138. print.
ISSN: 0021-9541.
DOCUMENT TYPE: Article
LANGUAGE: English

AB Previously, we demonstrated that the gastrin releasing peptide (GRP) induces cyclooxygenase-2 (COX-2) expression through a Rho-dependent, protein kinase C (PKC)-independent signaling pathway in fibroblasts (Slice et al., 1999, J Biol Chem 274:27562-27566). However, the specific role of heterotrimeric guanine nucleotide binding regulatory proteins (G-proteins) that are coupled to the GRP receptor in Rho-dependent COX-2 expression has not been elucidated. In this report, we utilize embryonic fibroblasts from transgenic mice containing double gene knock-outs (DKO) for Galphaq/11 and Galpha12/13 to demonstrate that COX-2 promoter activation by GRP requires Galphaq. Furthermore, we show that GRP-dependent COX-2 gene expression, as assessed by a COX-2 reporter luciferase assay, was induced in cells lacking Galpha12/13 but was blocked in cells that did not express Galphaq/11. GRP-dependent COX-2 promoter induction in Galphaq/11 deficient cells was rescued by expression of wild type Galphaq but blocked by inhibition of calcium signaling in calcium-free media or in cells treated with 2-aminoethoxydiphenylborate (2-APB). Co-stimulation of transfected Galphaq/11 deficient cells with GRP and thapsigargin (TG) induced the COX-2 promoter. Activation of endogenous Rho by expression of Onco-lbc or expression of Rho A Q63L resulted in COX-2 promoter activation in Galphaq/11 deficient cells. Inhibition of Rho by Clostridium botulinum C3 toxin blocked COX-2 promoter induction. Expression of Galphaq Q209L in the well-characterized fibroblast cell line, NIH3T3, induced the COX-2 promoter which was blocked by expression of C3 toxin. These results demonstrate that calcium signaling mediated by Galphaq and Rho play critical roles in GRP-dependent COX-2 expression in fibroblasts.

L3 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1
ACCESSION NUMBER: 2001:344364 BIOSIS
DOCUMENT NUMBER: PREV200100344364
TITLE: Activation of cellular invasion by trefoil peptides and src is mediated by cyclooxygenase- and thromboxane A2 receptor-dependent signaling pathways.
AUTHOR(S): Rodrigues, Sylvie; Nguyen, Quang-De; Faivre, Sandrine; Bruyneel, Erik; Thim, Lars; Westley, Bruce; May, Felicity; Flatau, Gilles; Mareel, Marc; Gespach, Christian (1); Emami, Shahin
CORPORATE SOURCE: (1) INSERM Unit U482, Hopital Saint-Antoine, 75571, Paris Cedex 12: gespach@st-antoine.inserm.fr France
SOURCE: FASEB Journal, (July, 2001) Vol. 15, No. 9, pp. 1517-1528. print.
ISSN: 0892-6638.
DOCUMENT TYPE: Article

LANGUAGE: English

SUMMARY LANGUAGE: English

AB We have investigated the possible functional relationships between cellular invasion pathways induced by trefoil factors (TFFs), src, and the cyclooxygenases COX-1 and COX-2. Pharmacological inhibitors of the Rho small GTPase (C3 exoenzyme), phospholipase C (U-73122), cyclooxygenases (SC-560, NS-398), and the thromboxane A2 receptor (TXA2-R) antagonist SQ-295 completely abolished invasion induced by intestinal trefoil factor, pS2, and src in kidney and colonic epithelial cells MDCKts.src and PCmsrc. In contrast, invasion was induced by the TXA2-R mimetic U-46619, constitutively activated forms of the heterotrimeric G-proteins Galphaq (AGalphaq), Galpha12, Galpha13 (AGalpha12/13), which are signaling elements downstream of TXA2-R. Ectopic overexpression of pS2 cDNA and protein in MDCKts.src-pS2 cells and human colorectal cancer cells HCT8/S11-pS2 initiate distinct invasion signals that are Rho independent and COX and TXA2-R dependent. We detected a marked induction of COX-2 protein and accumulation of the stable PGH2/TXA2 metabolite TXB2 in the conditioned medium from cells transformed by src. This led to activation of the TXA2-R-dependent invasion pathway, which is monitored via a Rho- and Galpha12/Galpha13-independent mechanism using the Galphaq/PKC signaling cascade. These findings identify a new intracrine/paracrine loop that can be monitored by TFFs and src in inflammatory diseases and progression of colorectal cancers.

L3 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 2

ACCESSION NUMBER: 2000:59882 BIOSIS

DOCUMENT NUMBER: PREV200000059882

TITLE: Oncogenic mutant of Galpha12 stimulates cell proliferation through cyclooxygenase-2 signaling pathway.

AUTHOR(S): Dermott, Jonathan M.; Reddy, M.V. Ramana; Onesime, Djamila; Reddy, E. Premkumar; Dhanasekaran, N. (1)

CORPORATE SOURCE: (1) Fels Institute for Cancer Research and Molecular Biology, Temple University School of Medicine, Philadelphia, PA USA

SOURCE: Oncogene, (Dec. 2, 1999) Vol. 18, No. 51, pp. 7185-7189. ISSN: 0950-9232.

DOCUMENT TYPE: Article

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Expression of the GTPase-deficient, activated mutant alpha-subunit of the heterotrimeric G protein G12 (Galpha12QL) leads to the neoplastic transformation of fibroblast cell lines. The mitogenic pathway regulated by Galpha12QL includes an extensive signaling network involving several small GTPases and various kinases. In addition, Galpha12QL has been shown to potentiate the serum-induced phospholipase-A2 activity in NIH3T3 cells. In the present study, we demonstrate that cyclooxygenase-2 (COX-2) pathway is involved in the mitogenic pathway activated by Galpha12QL. Expression of Galpha12QL and not Galpha13QL, stimulates the serum-induced release of arachidonic acid in NIH3T3 cells. Furthermore, expression of Galpha12QL or the stimulation of wild-type Galpha12 induces the expression of COX-2. Our results also indicate that the COX-2 inhibitor acutely disrupts the DNA-synthesis stimulated by Galpha12QL in NIH3T3 cells. These studies, for the first time, identify the crucial role of COX-2 in Galpha12-mediated-regulation of cell proliferation and suggest a role for prostaglandin-derived autocrine loop in Galpha12-mediated signaling pathways.

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<u>L3</u>	L2 same (GTPase mutant)	0	<u>L3</u>
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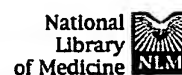
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<u>L3</u>	L2 same (GTPase mutant)	0	<u>L3</u>
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<u>L1</u>	COX-2	1698	<u>L1</u>

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☐ 1: Jpn J Cancer Res. 1997 Nov;88(11):1044-51.

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Inhibitory effect of NS-398, a selective cyclooxygenase-2 inhibitor, on azoxymethane-induced aberrant crypt foci in colon carcinogenesis of F344 rats.

PubMed
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Yoshimi N, Kawabata K, Hara A, Matsunaga K, Yamada Y, Mori H.

Department of Pathology, Gifu University School of Medicine.

Related
Resources

Prostaglandin E2, which is produced by cyclooxygenase (COX) during arachidonic acid metabolism, is considered to be related to colon carcinogenesis. Therefore, the effect of NS-398 (N-(2-cyclohexyloxy-4-nitrophenyl)methanesulfonamide), a COX-2 inhibitor, was examined in azoxymethane (AOM)-induced colon carcinogenesis in rats in this study. In the first experiment, groups 1-3 were treated with AOM (15 mg/kg, s.c.) 3 times at intervals of a week from 5 weeks of age. Groups 2 and 3 were respectively given 1 mg/kg and 10 mg/kg of NS-398 in 5% gum arabic aqueous solution 3 times per week by oral gavage during the experiment. Six weeks after the first exposure to AOM, aberrant crypt foci (ACF) were counted in the colonic mucosa of all rats. The mean occurrence of ACF per length in rats given 1 mg/kg b.w. or 10 mg/kg b.w. of NS-398 was reduced to 65.7% or 52.8%, respectively, of that in rats treated with only AOM. Levels of COX-2 mRNA expression in groups treated with AOM, regardless of NS-398, were slightly higher than that in the group treated with NS-398 alone as judged from reverse transcription-polymerase chain reaction analysis. In the second experiment, the effect of NS-398 at different times, i.e., during initiation and post-initiation, was examined. Treatment with NS-398 in both phases significantly inhibited the appearance of ACF. The results imply that NS-398 might have a chemopreventive potential.

PMID: 9439679 [PubMed - indexed for MEDLINE]

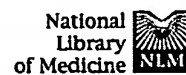
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☐ 1: Jpn J Cancer Res. 1997 Dec;88(12):1117-20.

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PubMed

Suppression of intestinal polyp development by nimesulide, a selective cyclooxygenase-2 inhibitor, in Min mice.

Nakatsugi S, Fukutake M, Takahashi M, Fukuda K, Isoi T, Taniguchi Y, Sugimura T, Wakabayashi K.

PubMed
Services

Cancer Prevention Division, National Cancer Center Research Institute, Tokyo.

Related
Resources

Nonsteroidal anti-inflammatory drugs (NSAIDs) suppress colon carcinogenesis in man and experimental animals. However, conventional NSAIDs inhibit both cyclooxygenase (COX) isoforms, COX-1 and COX-2, and cause gastrointestinal side-effects. Nimesulide, a selective inhibitor of COX-2, is much less ulcerogenic. We, therefore, examined its influence on the development of intestinal polyps in Min mice. Female Min mice at 4 weeks old were given 400 ppm nimesulide in their diet for 11 weeks. This treatment resulted in a significant reduction of the numbers of both small and large intestinal polyps, the total being 52% of that in untreated control Min mice. The size of the polyps in the nimesulide-treated group was also significantly decreased. The results suggest that nimesulide is a good candidate as a chemopreventive agent for human colon cancer with low toxicity.

PMID: 9473726 [PubMed - indexed for MEDLINE]

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☐ 1: Inflamm Res. 1997 Dec;46(12):496-502.

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Selective inhibition of cyclooxygenase-2 by NS-398 in endotoxin shock rats in vivo.

Futaki N, Takahashi S, Kitagawa T, Yamakawa Y, Tanaka M, Higuchi S.

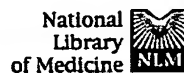
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Pharmaceutical Research Laboratories, Taisho Pharmaceutical Co. Ltd., Saitama, Japan.

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OBJECTIVE AND DESIGN: The role of cyclooxygenase (COX)-2 was examined using a rat endotoxin shock model and the potency and selectivity of NS-398, a COX-2 selective inhibitor in vitro, for COX-2 activity was examined in vivo. **MATERIAL:** Male Wistar rats (weighing 140-180 g) were used. **METHODS:** Lipopolysaccharide (LPS, 1 mg/kg, i.v.) was administered to rats (LPS-treated rats) and expression of COX-1 mRNA and COX-2 mRNA in the aorta and peripheral blood leukocytes was examined by RT-PCR. COX activity was assessed by measuring the plasma 6-keto prostaglandin (PG) F1 alpha, PGE2 and thromboxane (TX)B2 30s after administration of arachidonic acid (AA, 3 mg/kg, i.v.), NS-398 (0.3-100 mg/kg, p.o.) or indomethacin (0.3-3 mg/kg, p.o.) was administered 1 h before the AA injection. **RESULTS:** COX-2 mRNA was detectable in the aorta and peripheral blood leukocytes at least from 3 to 9 h after the LPS injection but not in non-LPS-treated rats. Plasma 6-keto PGF1 alpha, PGE2 and TXB2 levels after AA injection into LPS-treated rats were significantly enhanced compared to findings in non-LPS-treated rats. NS-398 showed significant inhibition of the increase in PGs in LPS-treated rats, the ED50 values being 0.35 mg/kg for 6-keto PGF1 alpha, 1.5 mg/kg for PGE2 and < 0.3 mg/kg for TXB2. NS-398 even at 100 mg/kg did not significantly suppress the increased PGs levels in non-LPS-treated rats. In contrast, indomethacin significantly inhibited plasma PGs levels after AA injection into LPS-treated rats and non-LPS-treated rats. The ED50 values in LPS-treated rats, determined by 6-keto PGF1 alpha, PGE2 and TXB2 production, were 1.0, 1.3 and 2.3 mg/kg and those in non-LPS-treated rats were 0.42, 0.24 and 0.93 mg/kg, respectively. **CONCLUSIONS:** In a rat endotoxin shock model, expression of COX-2 plays a role in an increase in COX activity. NS-398 showed preferential inhibitory effects on COX-2 activity in vivo. This approach is useful to directly analyze the inhibitory activity of NSAIDs for COX-1 and COX-2 in vivo.

PMID: 9459080 [PubMed - indexed for MEDLINE]



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☐ 1: Inflamm Res. 1997 Nov;46(11):437-46.

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In vitro and in vivo pharmacological evidence of selective cyclooxygenase-2 inhibition by nimesulide: an overview.

Famaey JP.

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Therabel Research S.A., Brussels, Belgium.

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Most available nonsteroidal anti-inflammatory drugs (NSAIDs) inhibit both the constitutive cyclooxygenase-1 (COX-1) and the inducible cyclooxygenase-2 (COX-2), resulting in inhibition of prostaglandin (PG) and thromboxane (TX) biosynthesis. The inhibition of COX-2 might be the cause of the favourable anti-inflammatory, analgesic and antipyretic effects of NSAIDs, whereas that of COX-1 might result in unwanted gastrointestinal, renal and possibly other side-effects. Nimesulide is a sulfonanilide compound with anti-inflammatory properties. Its pharmacological profile (better inhibition of PG synthesis in inflammatory areas than in gastric mucosa), suggested that it might be a selective inhibitor of COX-2. In several in vitro assays using either purified COX-2 and COX-1 preparations or cell preparations (both from animal and human origins) expressing COX-1 or COX-2, ten out of eleven different groups have demonstrated that nimesulide selectively inhibits COX-2. The COX-2/ COX-1 inhibitory ratio varies, according to the assay preparation, from about 0.76 to 0.0004 i.e. a 1.3 to 2,512-fold higher selectivity for COX-2 than for COX-1. Moreover, an in vivo whole blood assay performed on healthy volunteers demonstrated a significant fall in COX-2 PGE2 production without any effect on COX-1 TXB2 production in subjects treated with nimesulide (100 mg b.i.d. for 2 weeks) versus no effect on COX-2 PGE2 and an almost total suppression of COX-1 TXB2 in subjects treated with aspirin (300 mg t.i.d. for 2 weeks). Nimesulide can thus be considered a relatively selective COX-2 inhibitor. At the recommended dosage of 100 mg b.i.d., it is as effective an analgesic and anti-inflammatory agent as classical NSAIDs, and a well-tolerated drug with few side-effects according to large-scale open studies and a global evaluation of a large number of controlled and non-controlled comparative trials.

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☐ 1: Annu Rev Biochem. 1986;55:69-102.

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Arachidonic acid metabolism.

Needleman P, Turk J, Jakschik BA, Morrison AR, Lefkowitz JB.

Publication Types:

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PMID: 3017195 [PubMed - indexed for MEDLINE]

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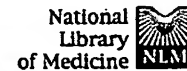
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☐ 1: Prostaglandins Leukot Essent Fatty Acids. 1998
Jun;58(6):421-4.

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Effects of lysine clonixinate on cyclooxygenase I and II in rat lung and stomach preparations.

Franchi AM, Di Girolamo G, de los Santos AR, Marti ML, Gimeno MA.

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Centro de Estudios Farmacologicos y Botánicos (CEFyBO), Consejo Nacional de Investigaciones Científicas y Técnicas (CONICET), Buenos Aires, Argentina.

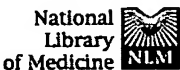


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Lysine clonixinate (LC) is a drug of antiinflammatory antipyretic and analgesic activity that produces minor digestive side-effects. This fact induced us to think that LC is possibly a weak COX-1 inhibitor. In order to investigate our hypothesis we inhibited cyclooxygenase activity with LC or indomethacin (INDO) in rat lung and stomach obtained from rats treated with lipopolysaccharide (LPS) and control rats. Rat lung preparations incubated with ^{14}C -arachidonic acid synthesise mainly PGE₂. LC at 2.5 and 4.1×10^{-5} M does not modify the basal production of PGE₂ (probably COX-1) but at 6.8×10^{-5} M significantly inhibited PGE₂ production (approximately 48.5% inhibition, $P < 0.001$). On the other hand, INDO at 10^{-6} inhibited the basal production of PGE₂ by around 73%. In LPS-treated rats, the production of PGE₂ was significantly higher than in the lungs of control rats, probably due to the induction of COX-2. The addition of LC at 2.7 and 4.1×10^{-5} M recovered the control values of PGE₂ inhibiting, probably only from COX-2 activity. LC at higher concentrations (6.8×10^{-5} M) and INDO 10^{-6} M inhibited PGE₂ formed by COX-2 and also partly by COX-1 activity.

PMID: 10189073 [PubMed - indexed for MEDLINE]

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☐ 1: J Physiol Pharmacol. 1998 Dec;49(4):501-13.

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Cyclooxygenase-2 selective and nitric oxide-releasing nonsteroidal anti-inflammatory drugs and gastric mucosal responses.

Takeuchi K, Suzuki K, Yamamoto H, Araki H, Mizoguchi H, Ukawa H.

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Pharmaceutical University, Yamashina, Japan. takeuchi@mb.kyoto-phu.ac.jp

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Occurrence of gastrointestinal damage and delayed healing of pre-existing ulcer are commonly observed in association with clinical use of nonsteroidal antiinflammatory drugs (NSAIDs). We examined the effects of NS-398, the cyclooxygenase (COX)-2 selective inhibitor, and nitric oxide (NO)-releasing aspirin (NCX-4016) on gastric mucosal ulcerogenic and healing responses in experimental animals, in comparison with those of nonselective COX inhibitors such as indomethacin and aspirin. Indomethacin and aspirin given orally were ulcerogenic by themselves in rat stomachs, while either NS-398 or NCX-4016 was not ulcerogenic at the doses which exert the equipotent antiinflammatory action with indomethacin or aspirin. Among these NSAIDs, only NCX-4016 showed a dose-dependent protection against gastric lesions induced by HCl/ethanol in rats. On the other hand, the healing of gastric ulcers induced in mice by thermal-cauterization was significantly delayed by repeated administration of these NSAIDs for more than 7 days, except NCX-4016. Gastric mucosal prostaglandin contents were reduced by indomethacin, aspirin and NCX-4016 in both normal and ulcerated mucosa, while NS-398 significantly decreased prostaglandin generation only in the ulcerated mucosa. Oral administration of NCX-4016 in pylorus-ligated rats and mice increased the levels of NO metabolites in the gastric contents. In addition, both NS-398 and NCX-4016 showed an equipotent anti-inflammatory effect against carrageenan-induced paw edema in rats as compared with indomethacin and aspirin. These results suggest that both indomethacin and aspirin are ulcerogenic by themselves and impair the healing of pre-existing gastric ulcers as well. The former action is due to inhibition of COX-1, while the latter effect may be accounted for by inhibition of COX-2 and mimicked by NS-398, the COX-2 selective NSAID. NCX-4016, despite inhibiting both COX-1 and COX-2, protects the stomach against damage and preserves the healing response of gastric ulcers, probably because of the beneficial action of NO.

PMID: 10069692 [PubMed - indexed for MEDLINE]

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PubMed

☐ 1: Prostaglandins Other Lipid Mediat. 1998
Aug;56(5-6):277-90.

[Related Articles, Links](#)

Lipopolysaccharide-induced expression of cyclooxygenase-2 in mouse macrophages is inhibited by chloromethylketones and a direct inhibitor of NF-kappa B translocation.

PubMed
Services**Abate A, Oberle S, Schroder H.**

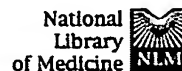
Department of Pharmacology and Toxicology, School of Pharmacy, Martin Luther University, Halle (Saale), Germany.

Related
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In macrophages, cyclooxygenase-2 (COX-2) is induced by cytokines, mitogens, or endotoxin. The present study investigates whether inhibitors of the nuclear transcription factor NF-kappa B affect lipopolysaccharide (LPS)-mediated expression of COX-2 mRNA, protein, and activity in the macrophage cell line J774.1A. The activation of COX-2 was assessed by measuring the accumulation of prostaglandin (PG) E2 by radioimmunoassay. Expression of COX-2 mRNA and protein was detected by Northern and Western blot analysis, respectively. In the absence of LPS, mouse macrophages did not express COX-2 and generated low amounts of prostaglandin (PG) E2. Treatment of J774.1A with LPS (0.1-30 micrograms/ml) caused expression of COX-2 protein and activity. Induction of COX-2 activity along with the induction of COX-2 mRNA and protein by LPS was attenuated by the serine protease inhibitors N-alpha-tosyl-L-phenylalanine chloromethyl ketone (TPCK) and N-alpha-tosyl-L-lysine chloromethyl ketone (TLCK). A cell permeable peptide and a direct inhibitor of NF-kappa B translocation, SN50, attenuated the accumulation of PGE2 in cell supernatant in a concentration-dependent manner. Our results show that induction of COX-2 by LPS in macrophages involves activation of NF-kappa B and point to a possible therapeutic use of protease inhibitors in inflammatory processes.

PMID: 9990673 [PubMed - indexed for MEDLINE]

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☐ 1: Carcinogenesis. 1998 Dec;19(12):2195-9.

[Related Articles, Links](#)

Entrez
PubMed

Meloxicam inhibits the growth of colorectal cancer cells.

Goldman AP, Williams CS, Sheng H, Lamps LW, Williams VP, Pairet M, Morrow JD, DuBois RN.

PubMed
Services

Department of Medicine, Vanderbilt University Medical Center, Nashville, TN 37232, USA.

Related
Resources

Cyclooxygenase-2 has been reported to play an important role in colorectal carcinogenesis. The effects of meloxicam (a COX-2 inhibitor) on the growth of two colon cancer cell lines that express COX-2 (HCA-7 and Moser-S) and a COX-2 negative cell line (HCT-116) were evaluated. The growth rate of these cells was measured following treatment with meloxicam. HCA-7 and Moser-S colony size were significantly reduced following treatment with meloxicam; however, there was no significant change in HCT-116 colony size with treatment. In vivo studies were performed to evaluate the effect of meloxicam on the growth of HCA-7 cells when xenografted into nude mice. We observed a 51% reduction in tumor size after 4 weeks of treatment. Analysis of COX-1 and COX-2 protein levels in HCA-7 tumor lysates revealed a slight decrease in COX-2 expression levels in tumors taken from mice treated with meloxicam and no detectable COX-1 expression. Here we report that meloxicam significantly inhibited HCA-7 colony and tumor growth but had no effect on the growth of the COX-2 negative HCT-116 cells.

PMID: 9886578 [PubMed - indexed for MEDLINE]

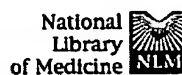
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#5	Search COX-2 inhibitor Field: Title/Abstract , Limits: Publication Date from 1990 to 1998	09:28:43	<u>255</u>
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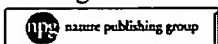
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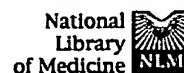
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PubMed☐ 1: Oncogene. 1999 Dec 2;18(51):7185-9.[Related Articles, Links](#)**Oncogenic mutant of Galpha12 stimulates cell proliferation through cyclooxygenase-2 signaling pathway.****Dermott JM, Reddy MR, Onesime D, Reddy EP, Dhanasekaran N.**PubMed
ServicesFels Institute for Cancer Research and Molecular Biology, Temple University
School of Medicine, Philadelphia, Pennsylvania, PA 19140, USA.Related
Resources

Expression of the GTPase-deficient, activated mutant alpha-subunit of the heterotrimeric G protein G12 (Galpha12QL) leads to the neoplastic transformation of fibroblast cell lines. The mitogenic pathway regulated by Galpha12QL includes an extensive signaling network involving several small GTPases and various kinases. In addition, Galpha12QL has been shown to potentiate the serum-induced phospholipase-A2 activity in NIH3T3 cells. In the present study, we demonstrate that cyclooxygenase-2 (COX-2) pathway is involved in the mitogenic pathway activated by Galpha12QL. Expression of Galpha12QL and not Galpha13QL, stimulates the serum-induced release of arachidonic acid in NIH3T3 cells. Furthermore, expression of Galpha12QL or the stimulation of wild-type Galpha12 induces the expression of COX-2. Our results also indicate that the COX-2 inhibitor acutely disrupts the DNA-synthesis stimulated by Galpha12QL in NIH3T3 cells. These studies, for the first time, identify the crucial role of COX-2 in Galpha12-mediated regulation of cell proliferation and suggest a role for prostaglandin-derived autocrine loop in Galpha12-mediated signaling pathways.

PMID: 10602471 [PubMed - indexed for MEDLINE]

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☐ 1: J Rheumatol. 1998 Nov;25(11):2279-81.

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Comment on:

- [J Rheumatol. 1997 Feb;24\(2\):243-5.](#)
- [J Rheumatol. 1997 Feb;24\(2\):246-8.](#)

Roles of COX-1 and COX-2.

PubMed
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McCormack K.

Publication Types:

- Comment
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PMID: 9818678 [PubMed - indexed for MEDLINE]

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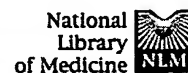
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☐ 1: Inflamm Res. 1999 Mar;48(3):133-8.

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Development of an in-vitro test system for the evaluation of cyclooxygenase-2 inhibitors.

Laufer S, Zechmeister P, Klein T.

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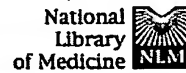
Department Drug Research, Merckle GmbH, Blaubeuren, Germany.

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OBJECTIVE AND DESIGN: The aim of our study was to establish an in-vitro test system, capable of fast and efficient screening of Cyclooxygenase-2 (COX-2) inhibitors. **MATERIALS:** Mononuclear cells were isolated out of human whole blood, in a one-step centrifugation procedure. **TREATMENT AND METHODS:** The time- and concentration-dependent induction of COX-2 expression in the blood monocytes (1×10^6 cells/ml) was evaluated by a kinetic profile. The optimal test conditions were fixed at an LPS concentration of 10 micrograms/ml and a 5 hour incubation time. The test compounds (10^{-5} to 10^{-8} mol/l) were set at $t = 0$ into the assay and were co-incubated for the whole period of COX-2 expression (5 hr). **RESULTS:** The following are representative examples of inhibitors with different distinct selectivity for COX-1/2. Indomethacin as a COX-1 selective compound inhibited PGHS-1 (IC₅₀: 0.002 microM) 200 times stronger than PGHS-2 (IC₅₀: 0.43 microM). Diclofenac had an almost equipotent efficacy on PGHS-1 (IC₅₀: 0.05 microM) and PGHS-2 (IC₅₀: 0.03 microM). NS-398 inhibited highly selective COX-2 (IC₅₀ PGHS-1: 10.75 microM vs IC₅₀ PGHS-2: 0.16 microM). **CONCLUSIONS:** The model reached the set targets with regard to the differentiation of COX-2 selective compounds, the reproducibility of results and practicability of the assay. In contrast to previous propounded theories, we could demonstrate, that mononuclear cells are not unusually sensitive to NSAIDs and apparently possess no further COX isoforms.

PMID: 10219655 [PubMed - indexed for MEDLINE]

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PubMed☐ 1: [Rowlinson SW, Crews BC, Lanzo CA, Marnett LJ.](#) [Related Articles, Links](#)

The binding of arachidonic acid in the cyclooxygenase active site of mouse prostaglandin endoperoxide synthase-2 (COX-2). A putative L-shaped binding conformation utilizing the top channel region.
J Biol Chem. 1999 Aug 13;274(33):23305-10.
PMID: 10438506 [PubMed - indexed for MEDLINE]

PubMed
Services☐ 2: [Marnett LJ, Rowlinson SW, Goodwin DC, Kalgutkar AS, Lanzo CA.](#) [Related Articles, Links](#)

Arachidonic acid oxygenation by COX-1 and COX-2. Mechanisms of catalysis and inhibition.
J Biol Chem. 1999 Aug 13;274(33):22903-6. Review. No abstract available.
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☐ 1: Biochem Soc Trans. 1990 Aug;18(4):503-7.

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Receptor-mediated activation of phospholipase A2 and arachidonic acid release in signal transduction.

Axelrod J.

PubMed
Services

Laboratory of Cell Biology, National Institute of Mental Health, Bethesda, MD 20892.

Publication Types:

- Review
- Review, Tutorial

PMID: 2177403 [PubMed - indexed for MEDLINE]

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File: PGPB

May 29, 2003

PGPUB-DOCUMENT-NUMBER: 20030100591

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030100591 A1

TITLE: Methods of treatment of uterine pathological conditions

PUBLICATION-DATE: May 29, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Jabbour, Henry Nicolas	Edinburgh		GB	

US-CL-CURRENT: [514/383](#); [514/16](#), [514/17](#), [514/406](#), [514/423](#), [514/573](#), [514/605](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw Desc	Image
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☐ 2. Document ID: US 20030083465 A1

L6: Entry 2 of 12

File: PGPB

May 1, 2003

PGPUB-DOCUMENT-NUMBER: 20030083465

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20030083465 A1

TITLE: Therapeutic and diagnostic methods and compositions based on Jagged/Notch proteins and nucleic acids

PUBLICATION-DATE: May 1, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Zimrin, Ann B.	Marriottsville	MD	US	
MaCiag, Thomas	Freeport	ME	US	
Pepper, Michael S.	Geneve	PA	CH	
Montesano, Roberto	Geneve		CH	
Wong, Michael	Pittsburgh		US	

US-CL-CURRENT: [530/350](#); [435/320.1](#), [435/325](#), [435/69.1](#), [536/23.5](#)

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments	Claims	KMC	Draw Desc	Image
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Jan 30, 2003

PGPUB-DOCUMENT-NUMBER: 20030022242
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030022242 A1

TITLE: Particles with improved solubilization capacity

PUBLICATION-DATE: January 30, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Anderson, David	Colonial Heights	VA	US	

US-CL-CURRENT: 435/7.1; 424/490

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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☐ 4. Document ID: US 20020177551 A1

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File: PGPB

Nov 28, 2002

PGPUB-DOCUMENT-NUMBER: 20020177551
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020177551 A1

TITLE: Compositions and methods for treatment of neoplastic disease

PUBLICATION-DATE: November 28, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Terman, David S.	Pebble Beach	CA	US	

US-CL-CURRENT: 514/12; 435/325, 530/350

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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☐ 5. Document ID: US 20020177152 A1

L6: Entry 5 of 12

File: PGPB

Nov 28, 2002

PGPUB-DOCUMENT-NUMBER: 20020177152
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020177152 A1

TITLE: COX 1-interacting proteins and use thereof

PUBLICATION-DATE: November 28, 2002.

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Wettstein, Daniel Albert	Salt Lake City	UT	US	

US-CL-CURRENT: 435/6; 435/189, 435/320.1, 435/325, 435/69.1

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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☐ 6. Document ID: US 20020022055 A1

L6: Entry 6 of 12

File: PGPB

Feb 21, 2002

PGPUB-DOCUMENT-NUMBER: 20020022055

PGPUB-FILING-TYPE: new

DOCUMENT-IDENTIFIER: US 20020022055 A1

TITLE: Composition and methods for improving integrity of compromised body passageways and cavities

PUBLICATION-DATE: February 21, 2002

INVENTOR-INFORMATION:

NAME CITY

Signore, Pierre E

Vancouver British Columbia

STATE COUNTRY RULE-47

CA

US-CL-CURRENT: 424/486

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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☐ 7. Document ID: US 6524795 B1

L6: Entry 7 of 12

File: USPT

Feb 25, 2003

US-PAT-NO: 6524795

DOCUMENT-IDENTIFIER: US 6524795 B1

TITLE: Diagnostics for cardiovascular disorders

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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KWIC	Draw Desc	Image
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☐ 8. Document ID: US 6451524 B1

L6: Entry 8 of 12

File: USPT

Sep 17, 2002

US-PAT-NO: 6451524

DOCUMENT-IDENTIFIER: US 6451524 B1

TITLE: Identification of disease predictive nucleic acids

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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☐ 9. Document ID: US 6433138 B1

L6: Entry 9 of 12

File: USPT

Aug 13, 2002

US-PAT-NO: 6433138

DOCUMENT-IDENTIFIER: US 6433138 B1

TITLE: Therapeutic and diagnostic methods and compositions based on jagged/notch proteins and nucleic acids

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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☐ 10. Document ID: US 6025194 A

L6: Entry 10 of 12

File: USPT

Feb 15, 2000

US-PAT-NO: 6025194

DOCUMENT-IDENTIFIER: US 6025194 A

TITLE: Nucleic acid sequence of senescence associated gene

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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☐ 11. Document ID: WO 200280927 A1 US 6440963 B1

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File: DWPI

Oct 17, 2002

DERWENT-ACC-NO: 2002-697104

DERWENT-WEEK: 200278

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TITLE: Method useful for treatment of neuromuscular dysfunction of lower urinary tract, e.g. dysuria, incontinence, enuresis and micturition disorders, involves use of selective inhibitors of cyclooxygenase-2 isozyme

Full	Title	Citation	Front	Review	Classification	Date	Reference	Sequences	Attachments
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File: DWPI

Apr 2, 2003

DERWENT-ACC-NO: 2002-055338

DERWENT-WEEK: 200325

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TITLE: New 2-fluorobenzenesulfonyl derivatives are cyclooxygenase2 inhibitors useful for treating inflammation, inflammation related disorders and cancer

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